diastolic sound, the accentuation and rhythm may be that of the *bruit de galop*. There is, however, a diastolic murmur heard in the dilated heart of children which is presumably due to that dilatation, and is indistinguishable from the diastolic sound of mitral stenosis. It may be best heard at the impulse or just outside it, or over the right ventricle in the third and fourth or fourth and fifth intercostal spaces. In one position it is probably produced in the left ventricle, in the other in the right. In the same heart it may sometimes be best heard in one position, sometimes in the other, and then probably points to general dilatation.

"Whether such a murmur indicates mitral stenosis or dilatation of the heart is of some importance. Adherent pericardium is the most common cause of a dilated heart in childhood, and death from such a cause is far more common than from mitral stenosis. Thus the post-mortem reports at Guy's for seven years showed that there were only 3 deaths from mitral stenosis of the age of fifteen years and under, while there were 13 deaths from cardiac disease with adherent pericardium, in which valvular disease was either absent or there was merely slight thickening of the mitral valves. Apparently very few children with adherent pericardium survive the age of fifteen, while mitral stenosis is not uncommon in young adults. This suggests that the immediate prognosis is considerably worse in the former affection. A diastolic sound, therefore, indicating persistent dilatation of the heart, is probably of far more serious import than one pointing to mitral stenosis."—*British Medical Journal*, 1894, No. 1739.

SUDDEN INSENSIBILITY AFTER EXTREME MUSCULAR EXERTION.

POORE (London) records the following case (*Lancet*, 1894, No. 3687): A strongly built man, aged twenty-one, was admitted to University College Hospital in an unconscious and irritable condition on February 25th. He had travelled to London, 120 miles, on the previous day to run in a ten-mile race, for which he had been training for some time. He felt out of sorts when the race began, but did a good time of 66½ minutes across very heavy country. During the race he did not perspire as much as he ought to have done, and afterward when he went to the dressing-room he felt very thirsty and drank off about a pint and a half of cold water. After this he became ill, did not recollect dressing himself, and, in fact, remembered nothing more until he found himself in the hospital next evening. His friends state that the race was finished about 5.45 P.M., and after this the patient dressed himself with very little assistance, but he did not seem well; he appeared "over-run." Just after leaving the dressing-room with his friends he "fainted." They gave him some brandy, but, as he became unconscious and helpless, they carried him to a neighboring house and laid him down on the floor. Here he lay quite still, with semi-closed eyes, occasionally groaning as though in pain. His breathing was stertorous, and his pupils are said to have been dilated equally and his teeth clenched. He continued thus until 7 P.M., when he began to have convulsions, which appear to have been epileptiform in character. They came on with intervals of one or two minutes, in which the breathing was stertorous and the patient was quite quiet. A considerable quantity of saliva ran from his mouth, but he did not bite his tongue. At 8 P.M. the convulsions became so violent that it was found necessary to restrain the patient, and at 11 P.M. he was given a hypodermatic injection of morphine. This controlled the fits, and he lay quiet during the rest of the night, but was quite unconscious. All next day, February 25th, he remained in the same condition, and in the evening was brought up to hospital. On admission he was in a state of extreme cerebral irritation. The pupils were equal and semi-dilated; the breath foul; the man would not answer questions or put out his tongue; the pulse was regular, full, and bounding; the urine normal. Attempts to feed by the nasal tube had to be given up on account of the violence of the patient. He then sat up in bed, seemed quite rational, drank a pint and a half of milk, and took three grains of calomel. He was restless, but quite conscious during the night. There was some headache on the following day, and the patient left the hospital on March 4th, no bad symptoms having developed in the interval.

In his remarks on the case, Poore considers as probable factors in the causation, waste products circulating in the blood, hyperpyrexia from suspension of sweating, and possibly sudden cerebral anæmia from drinking a large quantity of cold water after the race.

DIPHTHERIA OF THE GENITALS FOLLOWED BY PARALYSIS.

GAYTON (London) mentions the case of a well-nourished girl of four years admitted to hospital with membranous diphtheria of the pudendum and groins. There was slight albuminuria and no fever. After three weeks the local condition was healed, but the albuminuria persisted. A week later strabismus was noted, and in the course of a few days paralysis became almost universal and eventuated in death by asphyxia. There had never at any time been any affection of the fauces or naso-pharynx. It is noted that out of 2733 cases which have come under observation, in only 6 were the genitalia affected.—*Lancet*, 1894, No. 3691.

THE PATHOGENESIS AND TREATMENT OF THE DELIRIUM OF PNEUMONIA.

AT the Eleventh International Medical Congress recently held at Rome, ROBERT (*Le Mercredi Médical*, 1894, No. 19, p. 230) dilated upon the gravity of the delirium of pneumonia, especially by reason of its inhibitory influence upon the tracheo-bronchial reflex centre, which, it has been demonstrated, is situated in the dorso-lateral portion of the pneumogastric centre. It was pointed out that the cerebral disturbances that appear in the course of an attack of pneumonia do not have a common pathogenesis. The most rational explanation is to be found in the action of various toxins or toxalbumins. The delirium that occurs during the period of engorgement and red hepatization is probably due to hyperæmia of the cerebral membranes; that occurring during the period of gray hepatization, to the meningeal complications resulting from secondary infection; that occurring during the period of defervescence, to cerebral anæmia or perhaps to passive congestion. The delirium of active cerebral hyperæmia is characterized by great agitation and marked insomnia; the face is red, the sclerotics injected, the pupils contracted, the pulse as a rule frequent and strong. Therapeutically, anti-